Screening might be done as an extension of the already operational screening procedures such as those for breast or cervical cancer. However, it is the over-55 age group who are at the greater risk and thus screening for ovarian cancer in that age group could be planned separately. Screening by clinical assessment could be provided at Well Women's Clinics for the 40–60 age group, Menopausal Clinics for the 45–55 age group and Family Planning Clinics for the under 45s.

Tumour markers in the blood might provide a screening tool but at present they are best used in following remission and recurrence in advanced disease. Perhaps in the future they may hold promise for early detection and screening.

Ultrasound might provide the most accurate screening method, but cost and shortage of manpower would be drawbacks at present. Combined breast and pelvic ultrasound screening might be more cost effective. The use of grey scale display and modern ultrasound equipment permits the detection of ovarian lesions as small as 1 cm in diameter which would be undetectable clinically (Meire *et al.* 1978), although no study has yet shown that detection at this size leads to better results. With increasing experience in ultrasound diagnosis, various criteria have been drawn up to help differentiate benign from malignant lesions with an accuracy of 70–90% (Meire *et al.* 1978, Samuels 1975, Kobayashi 1976).

Whatever form of screening for ovarian cancer is used eventually, there is already a case for a pilot study to compare clinical and ultrasound detection of early ovarian cancer.

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Foreign body perforation of a jejunal diverticulum[1]

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A case of perforation of a jejunal diverticulum by a fish bone is reported. This is only the fourth case in the world literature. The complications of jejunal diverticula and the causes of foreign body perforation of the bowel and Meckel's diverticula are reviewed.

[1]Case presented to Clinical Section, 13 November 1981. Accepted 12 February 1982

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Case report

A 60-year-old engineer for North Thames Gas presented as an emergency with a 15-hour history of severe abdominal pain. The pain was lower abdominal, had an acute onset and woke him from sleep. He had been nauseated but had not vomited. There were no other systemic symptoms. Previously he had suffered from multiple sclerosis (1967) and had been found to have duodenal and jejunal diverticula (1971) following the investigation of epigastric pain.

On examination there was tenderness across the lower abdomen with guarding and rebound tenderness in the right iliac fossa. Otherwise he was apyrexial and all other systems were normal. A diagnosis of acute appendicitis was made and he was prepared for theatre.

On opening the abdomen through a grid iron incision, purulent generalized peritonitis with a normal appendix was found; the appendix was removed and the wound closed. The abdomen was reopened through a right paramedian incision and a midjejunal diverticulum was found to be perforated by a fish bone some 2 cm in length. The fish bone was removed, the perforation, which was acute and clean, was oversewn, the abdominal cavity lavaged, drained and closed.

Recovery was complicated by a left lower lobe pneumonia and a haematemesis from an acute gastric ulcer and multiple gastric erosions. These were treated conservatively and settled. On questioning, the patient remembered a meal of cod 48 hours before admission.

Discussion

It is not known with certainty whether jejunal diverticula are congenital or acquired. Meckel's diverticulum is a congenital structure (persistent vitello-intestinal duct) affecting some 2% of the population, and other diverticula have been found in embryos (20–30 mm) (Bremer 1944). However, the favoured theory for the formation of jejunal diverticula is similar to that postulated for sigmoid diverticular disease; namely, an outpouching of mucosa at the entry of a blood vessel (locus minoris resistantiae) (Baskin & Mayo 1952) (Figure 1). Indeed, the longitudinal muscle in the jejunum is deficient at the junction of the bowel with its mesentery. However, the duodenum has no such deficiency and diverticula are more common here (Philips 1953). It has also been suggested that these structures are in fact pulsion diverticula caused by uncoordinated bowel contraction (Edwards 1936).

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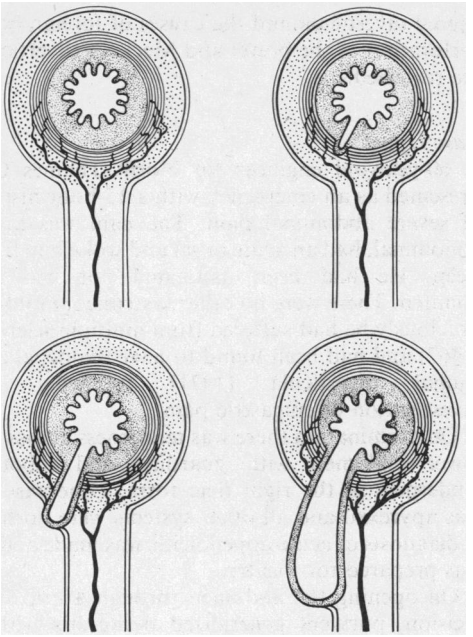

Figure 1. Diagram illustrating the formation of a diverticulum

Jejunal diverticula were first described by Sir Astley Cooper in 1804. They consist of mucosa, submucosa and peritoneum and are situated on the mesenteric side of the bowel. They are usually 1–4 cm in diameter but are found up to 8–9 cm in diameter; 70% are found at post-mortem, 18% at laparotomy and 12% on barium follow through (Benson *et al.* 1943). The overall incidence is 1.3% (Rosedale 1935). No complications occur in 61% of patients, 29% have mild abdominal discomfort and 10% require surgical intervention (Baskin & Mayo 1952). Table 1 lists the complications which may be related to the thin wall with its nearby artery or to the fact that the diverticulum is a bywater on the gut. However, compared to the colon, the small bowel diverticulum is usually wide mouthed and thus perhaps tends not to collect food so readily; it is also normally sterile.

Table 1. Complications of jejunal diverticula (Orr & Russell 1951)

Acute diverticulitis—perforation
—adhesion
Haemorrhage
Malabsorption/vitamin B_{12} deficiency
Obstruction—volvulus, torsion, adhesion
—dysfunction
Traumatic rupture
Foreign body perforation
Concretion formation
(Malignancy)

Table 2. Foreign bodies and the intestine

Perforate	Pass
Fish bones	Open safety pins
Animal bones	Pins
Toothpicks	Nails
	Needles
	Glass

Foreign bodies which penetrate the gut tend to be non-metallic and of animal or vegetable origin (Ashby & Hunter-Craig 1967) (Table 2). Sharp metallic objects generally pass through; this may be because they present a different clinical problem. Swallowing a metallic object is more dramatic, patients present earlier and are observed for potential perforation. The object's progress is followed by serial X-ray examinations and failure to pass leads to early intervention.

Non-metallic foreign bodies present late with complications. These can be peritonitis, abscess formation or an inflammatory mass either intra-abdominal, situated in the abdominal wall or in a hernial sac. Sites of perforation have been reported in the stomach, duodenum, jejunum, ileum, caecum, colonic flexures, sigmoid colon and rectum. There is an apparent predisposition to perforation in people with dental plates and alcholism, both of which decrease intra-oral sensation (Ginzburg & Beller 1927). Perforation of Meckel's diverticulum forms a good model for perforations of diverticula, and from Table 3 it may be noted that they are mainly due to fish bones (Rosswick 1965).

Table 3. Perforations of Meckel's diverticulum (from Rosswick 1965)

Fish bones	28
Wood splinters	6
Needles	4
Fruit stones	3
Tomato skins	3
Chicken bones	1
Cabbage stalk	1
Ascaris	1
Grape seeds	1
Jewellery	1
Artichoke spine	1

The suggested mechanisms of perforation are:

(1) The foreign body produces an area of necrosis and gradually works its way through. Usually there are two areas of necrosis, one at each end of the foreign body.

(2) An area of necrosis results and secondary perforation occurs after the foreign body has passed on.

(3) A small sharp foreign body is pushed straight through the bowel wall with minimal necrosis (Ginzburg & Beller 1927).

Fish bones are presumably effective perforators because they tend to be sharp at both ends, are frequently swallowed and, perhaps because of their curve, they tend to lodge in the bowel.

This case is only the fourth case of perforation of a jejunal diverticulum by a foreign body to be described in the literature. One case was caused by a 3 cm length of steak bone (Navarre & Schmidt 1958), one by a 2 cm fish bone (Fidler 1972) and the third by a vegetable stalk (Shaw 1980). This low incidence of perforation by foreign bodies is in marked contrast to Meckel's diverticulum where it is a well recognized complication (Rosswick 1965), in spite of the incidence of diverticula being similar in these two sites. It is perhaps accounted for by the wide neck which allows the relatively easy influx and egress of food in jejunal diverticula.

Perforations are usually secondary to diverticulitis (Babcock *et al.* 1976), but may be due to trauma or even slow-release iron tablets (Ingold 1977). Often there is no previous history of jejunal diverticulosis and the diagnosis is made at laparotomy.

Surgical treatment ideally should remove the affected segment, but if a very large resection is required just the perforated segment may be removed. An acute perforation may simply be oversewn and, if necessary, the diverticulum invaginated or an omental patch applied.

Acknowledgment: I should like to thank Mr C C Ware for permission to report this case, and Mr S Ganley for Figure 1.

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Meleney's progressive synergistic bacterial gangrene due to subcutaneous end-ileostomy perforation, with delayed plastic reconstruction[1]

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Meleney's progressive synergistic bacterial gangrene is a rare form of gangrene. It usually affects the trunk, though occasionally the limbs and, once controlled, leaves a large exposed area which necessitates plastic reconstruction. A case is now reported of a subcutaneous end-ileostomy perforation, leading to Meleney's synergistic gangrene, in a man on whom delayed plastic reconstruction was performed to cover the bare area and refashion a new end-ileostomy faceplate.

Case report

A 48-year-old Caucasian man was admitted to his local hospital complaining of malaise and increasing pain around his end-ileostomy, created in 1978 at panproctocolectomy for ulcerative colitis. On examination he was pyrexial with duskiness, blistering and tenderness around his stoma, but no intra-abdominal signs. He was treated by intravenous rehydration, penicillin, gentamycin and metronidazole. However, the area affected extended dramatically over 24 hours, and a diagnosis of Meleney's progressive synergistic bacterial gangrene was made. Early bacteriology showed only *Escherichia coli* and *Streptococcus faecalis*.

The patient was transferred to Westminster Hospital for hyperbaric oxygen treatment but, despite three periods of treatment, by the third day of hospitalization the affected area had extended to 50×20 cm around the end-ileostomy. Consequently operative excision of the affected area with a margin of normal tissue and down to the intact deep fascia was performed; an inferior perforation of the stoma was noticed just superficial to the deep fascia. Postoperative soiling from the stoma caused an abscess to form in the

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